A case report makes you a world's expert for a day. Why not record it!

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As yew like it

25 year-old man found comatose in a park

Hypotensive, 30-250 bpm, afebrile, responsive to deap pain; he was intubated

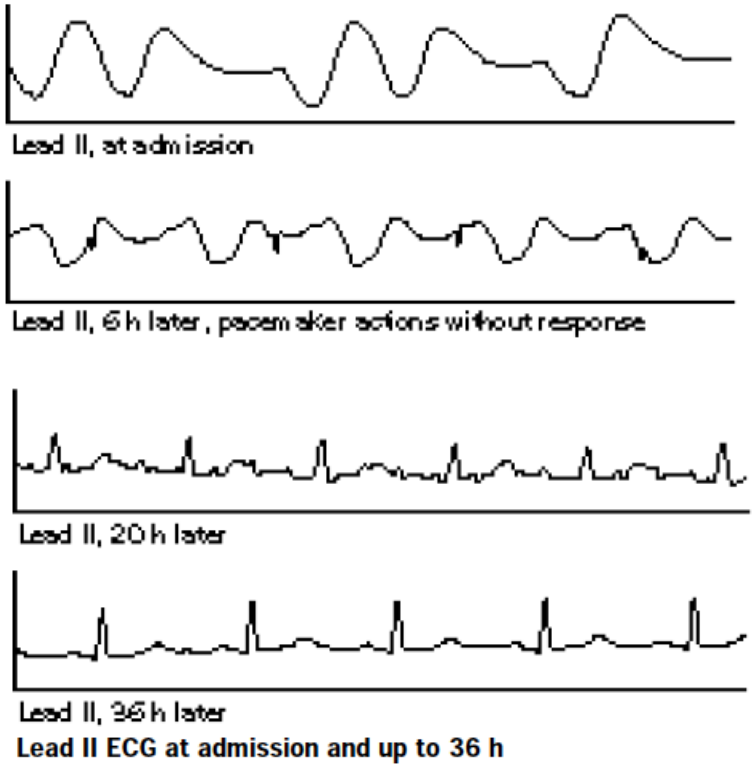
Naloxone had no effect

PaO2 660 mm Hg, PaCO2 25 mm Hg, pH 7.29 HCO3 11
Na 133, K 2.9, Cl 97 (mmol/L), PCr 125 µMol/L

(AL = 25, PaO2 suggests FiO2 100%, hypokalemia unclear)

Almost continuous resuscitation for 8 h. See pacemaker spike

ECG interpretation?



Lead II ECG at admission and up to 36 h

The nurse went through his pockets and found these

(Taxus baccata)

Pilz et al. Lancet 1999

See also:
Lancet 1836

Deiterpenoid alkaloids interfere with sodium and calcium channels

Called because of diabetic ketoacidosis

54 year-old patient released from Cancer Center after treatment for synovial sarcoma. He has DM-type 2. He had received irratiation, doxorubicin and ifosfamide. We were called because of increased creatinine and hypokalemia.

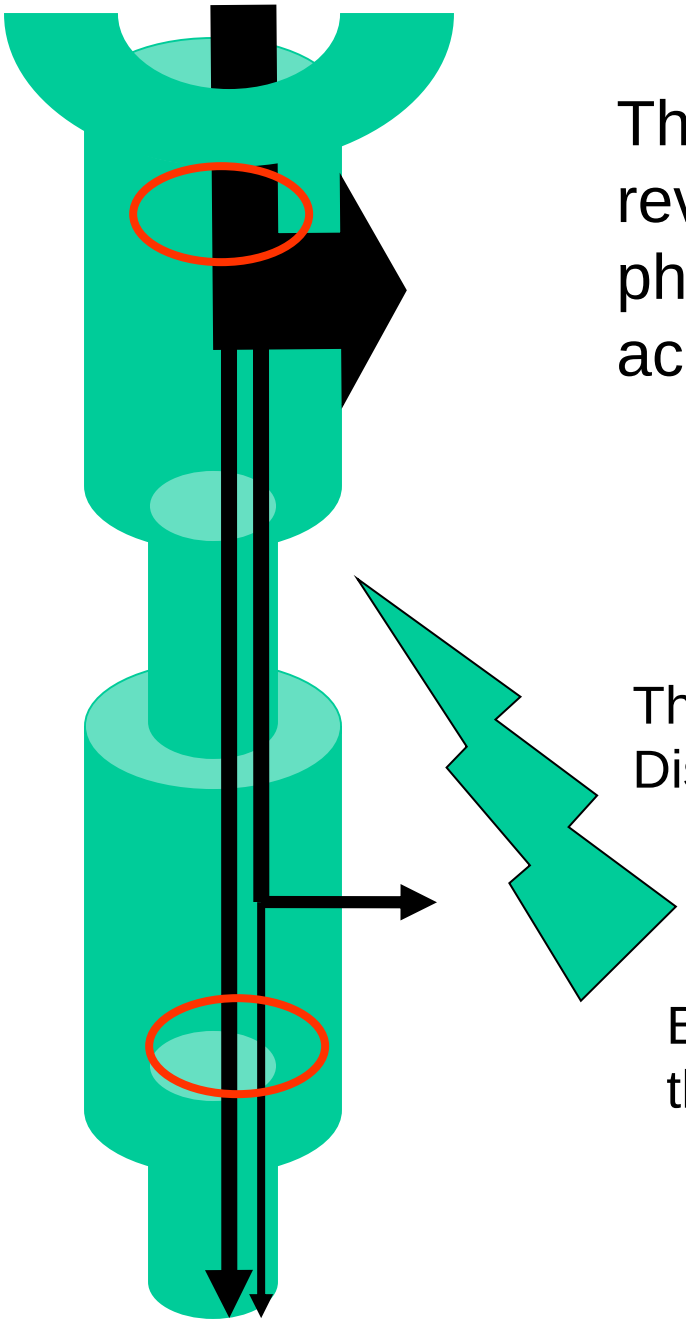
		Admission
Serum (S)		
Na	mmol/l	160
K	mmol/l	2.3
Cl	mmol/l	140
Glucose	mg/dl	300
Creatinine	mg/dl	3.69
Phosphate	mmol/l	n. d.
pH		7.24
pCO_2	mmHg	12
pO_2	mmHg	122
HCO_3	mmol/l	5
AG	mmol/l	15
Urine (U)		
Na	mmol/l	30
K	mmol/l	12
Cl	mmol/l	40
Glucose	mg/dl	1000
Phosphate	mmol/l	n. d.[c]
Ketone		negative
pCO_2	mmol/l	16.1
pH		5
FE HCO_3[a]	%	ca. 0.4
UAG[b]	mmol/l	+2.0

[a] FE, fractional excretion; FE $HCO_3^- = U[HCO_3^-] \times S[Cr]/S[HCO_3^-] \times [Cr] \times 100$

[b] UAG, urinary anion gap; $UAG = (U[Na^+] + U[K^+]) - U[Cl^-]$

[c] n.d., not determined.

Ifosfamide nephrotoxicity



Three months later, the creatinine had reverted to 1.4 mg/dl, the glycosuria and the phosphaturia had abated, and the metabolic acidosis was gone.

The usual: H&P, lab values, UAG, Prox RTA, Distal RTA. Probable cause - should go away

Bicarbonate requirement much greater than 1 mmol/kg/day

We did „nothing“

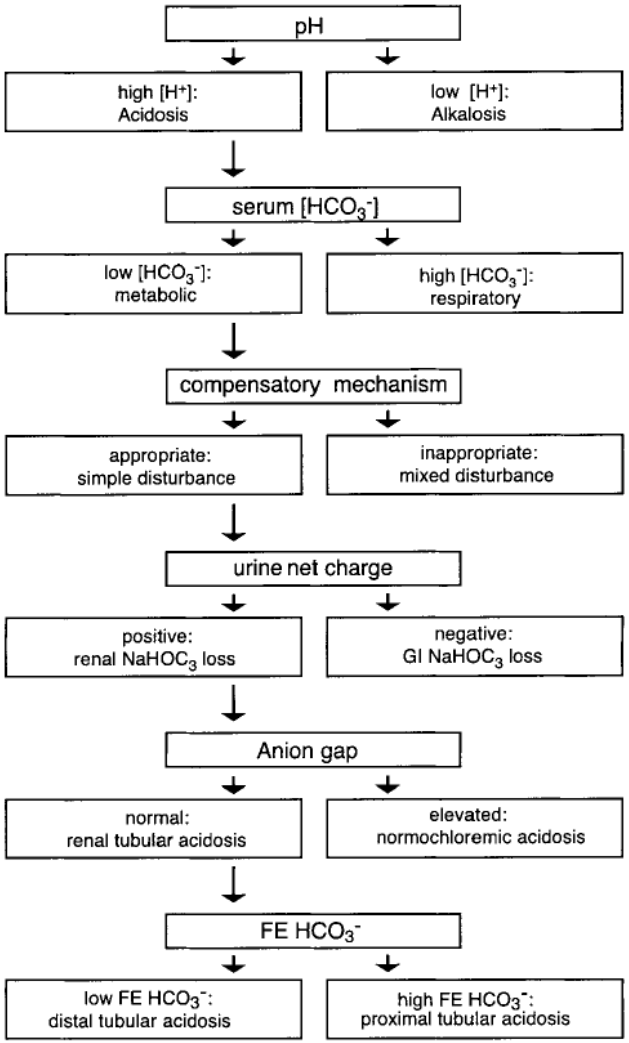


Diagram for evaluating metabolic acidosis

	Day 2
Serum (S)	
Na	162
K	4.0
Cl	132
Glucose	307
Creatinine	4.39
Phosphate	0.6
pH	7.46
pCO_2	20
pO_2	87
HCO_3	14
AG	11
Urine (U)	
Na	75
K	34
Cl	86
Glucose	1000
Phosphate	1.47
Ketone	n. d.
pCO_2	n. d.
pH	7
FE HCO_3[a]	n. d.
UAG[b]	-23

Lehrich et al. NDT 1999

Souring on dialysis

49 year-old dialysis patient with history of anorexia nervosa, started doing this:
She takes CaCO3, cinacalcet, EPO, iron, vitamins, and candesartan. Makes <100 ml urine/day

Table 1 | Excerpts from arterial blood gas and electrolytes at four different time points

	Before dialysis	After	24 h later	Before next dialysis
pH	7.19	7.47	7.33	7.09
$PaCO_2$ (mm Hg)	28	35	41	42
HCO_3^- (mmol/l)	10	25	21	12
Na^+ (mmol/l)	132	139	137	139
Cl^- (mmol/l)	95	101	98	102
AG (mmol/l)	27	13	16	25

Abbreviation: AG, anion gap.

Methanol, ethylene glycol, ketones, and salicylates were negative.
Acid-base disturbance?

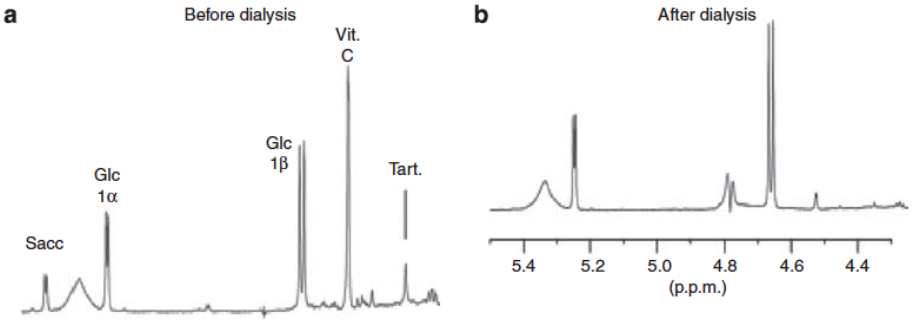


Figure 1 | Sections of the 1H nuclear magnetic resonance spectra showing the region of interest (5.5–3.5 p.p.m.) are shown. (**a**) The spectrum before dialysis shows a saccharose peak, two glucose peaks, a vitamin C peak, and a tartrate peak. (**b**) The spectrum after dialysis shows disappearance of the saccharose peak, vitamin C peak, and the tartrate peak. Glc1α, signal of proton in C-1α position; Glc1β, signal of proton in C-1β position; Sacc, saccharose/sucrose; Tart., tartrate; Vit. C, vitamin C.

Ahoj-Brause candy contains tartaric acid (Weinsäure).

The candies explained the peaks

Elitok et al. Kidney Int 2010

Dialysis patient develops metabolic alkalosis

77 year-old dialysis patient with dyspnea for 1 week

She has MPO-ANCA vasculitis; cylclophosphamide and prednisone.

pH 7.44, PaO2 66 mm Hg, PaCO2 39 mm Hg, HCO3 26 mmol/L

She receives more cyclophosphamide, prednisone, and plasmapheresis (PPH) with FFP to remove MPO ANCA. She receives 3 PPH treatments.

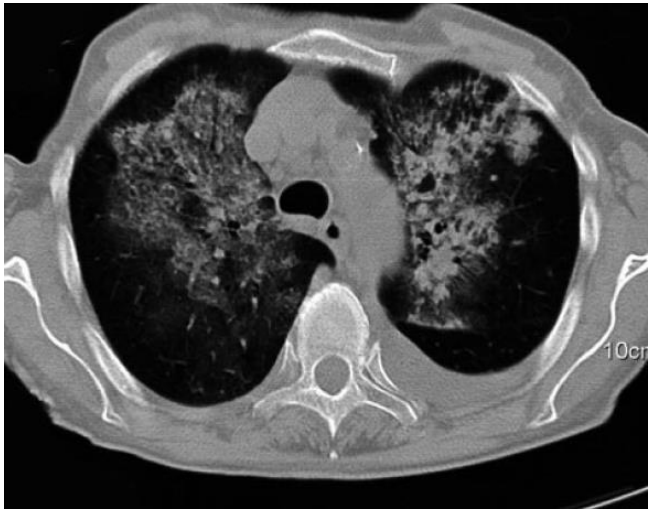


Table 1. Blood gas values during the initial plasmaphereses (PPH)

Time	pH	$PaCO_2$	PaO_2	HCO_3
Before PPH	7.41	53	54	33
1/2 PPH	7.41	53	52	33
After PPH	7.44	54	50	35
After haemodialysis	7.37	62	84	35

Where does this metabolic alkalosis come from?

Table 2. Blood gas values during plasmaphereses (PPH) with 5% albumin containing no sodium citrate

Time	pH	$PaCO_2$	PaO_2	HCO_3
Before PPH	7.43	45	75	29
1/2 PPH	7.35	39	82	21
After PPH	7.37	50	64	28
After haemodialysis	7.41	42	74	26

FFP is anticoagulated with sodium citrate ($C_6H_5O_7Na_3$). Each molecule yields $3NaHCO_3$.

Dialysis patients cannot excrete bicarbonate. Thus, they develop metabolic alkalosis with PPH.

Von Vietinghoff et al. NDT 2005

A glowing report on dialysis

36 year-old woman on dialysis. She is found to have papillary carcinoma of the thyroid.

Tratment with radioactive iodine (^{131}I) was initiated (2952 MBq). Patients with normal renal function excrete this material within 50 h.

But who knows about dialysis patients (no-one)?

In Germany, authorities „go crazy“ about this stuff.

We were called by nuclear medicine: “Our patient is „still glowing!“ Of course they called on Sunday afternoon.

Can radioactive materials be dialyzed off?

Is there any danger to nursing or medical personal?

What about the equipment? Does it have to be thrown away?

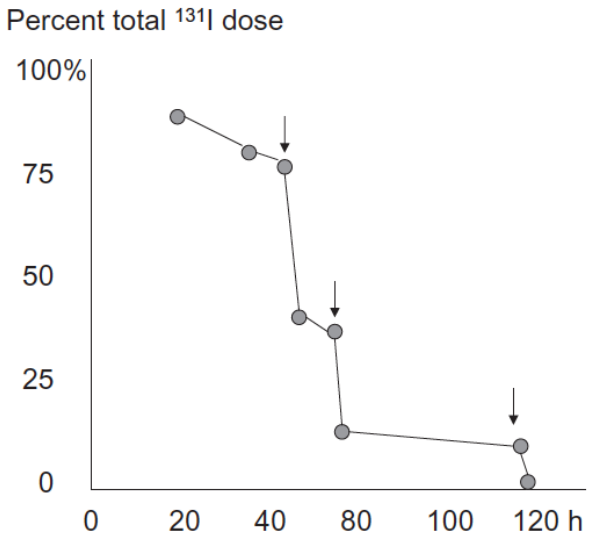


Fig. 1. The percentage total ^{131}I dose exhibited by the patient is shown. She was dialysed at 44, 76 and 120 h (arrows). The prompt decline in radioactivity is evident.

Iodide is like the chloride space; it should „dialyse off“.
The machinery is not encumbered.
The dialysate was gathered in containers and delivered to nuclear medicine. Medical personnel in Germany can be exposed to 1000 µS/per year. Our personnel received far less.

Stabroth and Luft. NDT 2005

Hyperkalemia mystery case

50 year-old woman develops hyperkalemic episodes

She had bad hypertension and only a single kidney

Her creatinine was 2.5 mg/dL

For unexplained reasons, she shows up in ERs with hyperkalemia. CrCl 50 ml/min, 24 h UK 82 mmol.

At presentations her K is 7-8 mmol/L. Resonium does not help.

The episodes are repetitive; she is dialyzed because of K. She even develops cardiac arrest and a pace-maker is implanted

The episodes occur sometimes 3xweekly but then about 1 per month; dialysis is perfomed.

SCN4A, KCNJ2 and other genes were sequenced.

What could this be? A national expert postulated that she had erythroptosis (I had no idea about that either).

We were all clueless.

When this patient comes into an ER, what needs to be (desparately) measured in this patient? The stuff in the table was not helpful.

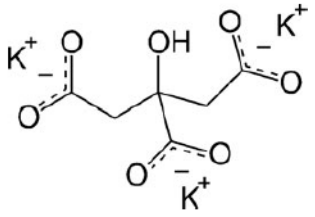
Parameter/date	11.12.06	8.1.07	12.2.07	21.2.07	3.3.08	7.4.08
Leukocytes (/nl)	8.91	5.95	6.7	8.11	4.3	4
Erys (/pl)	3.75	4.06	4.39	4.13	2.8	3.7
Hb (g/dl)	11.9	12.6	13.5	12.9		10.9
PCV (%)	35.9	37.3	40.9	38.1	25.8	32.6
MCV (fl)	95.7	91.9	93.2	92.3	91	89
MCH (pg)	31.7	31	30.8	31.2	31	30
Thrombo (/nl)	246	260	271	282	242	293
Hypochr. Erys (%)	0.2	0.2	0.1	0.1		
RET-He (pg)	34.4	33.2	34.3	34.6	33.5	28.7
Retics (%)	2.7	3.2	3.3	3	1.66	1.58
Creatinine (mg/dl)	1.34	1.45	1.49	1.49	3.4	4.9
Urea (mg/dl)	56	20	39	54		87
K^+ (mM)	4.6	4.5	>8.0	6.3		3.1
Ca^{2+} (mM)	2.19	2.12	2.26	2.38	2.2	2.2
PO_4^{2-} (mM)	0.84	0.94	0.55	0.54	1	1.6

She needed an acid-base/blood-gas) work-up at every ER admission and this was not done.

She had enough renal function to eliminate K normally and took no interfering drugs

One day, she vomited in the ER and I was called

The vomit K was 190 mmol/L, Cl was 114 mmol/L, Na 48 mmol/L. Citrate was >5 g/L.

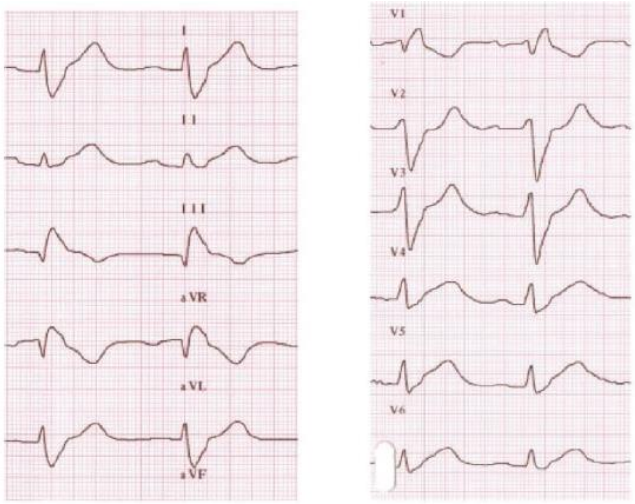


Kalinor Brause

Morgenstern et al NDT 2009

Lactate in a *Laubenpieper* (there is no adequate translation)

47 year-old „unemployed“ patient develops edema, diarrhea, and failure to thrive. He resides in a so-called „garden colony“. He denies excessive alcohol intake (Berliner scale). BP 115/60 mm Hg, HR 96/min, RR 36/min. Neckveins distended – edema to sacrum. Eye movements OK. Ph 7.3, PaO_2 105, $PaCO_2$ 20 (all mm Hg), Na 123, K 6.1, Cl 87, HCO_3 10 (all mmol/L). Glucose 5.6 and lactate 14 mmol/L.



Are life-threatening features present? Are initial therapeutic steps required?

Metabolic acidosis, increase in AL, explained by lactate, not in shock (type A versus type B lactic acidosis.

Hyperkalemia warrants prompt treatment.

What is driving this lactic acidosis?

O_2 Sat was normal. Mixed veonous PO_2 was 63 mm Hg.

Catherization confimed high-output heart failure.

B1 values were not zero but were low in this patient.

Derer et al. NDT 2005

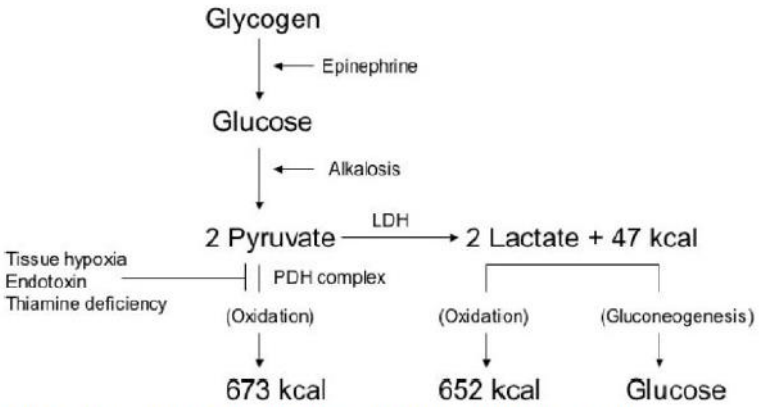


Fig. 2. The important features of glucose and lactate metabolism are given.

Recurrent paroxysms of metabolic acidosis in a hemodialysis patient

56 year-old dialyis patient commonly came into treatments with

Ph 7.14, PaO2 124, PaCO2 27, HCO3 9 (mmol/L).
Na normal, Cl elevated, so that the AL was always normal.

She dialyzed 5 h againts a 35 mmol/L dialysate. End Treatment: pH 7.4, PaO2 120, PaCO2 34 mg Hg.

Then, she comes in with pH 7.07, PaCO2 20 mm Hg, and HCO3 5 mmol/l. Cl was elevated so that there was no „anion gap“.

Similar eposides (pH 7.07, PaCO2 20 mm Hg, HCO3 5 mmol/L with no AG and elevated chrloide values) occurred repetitively.

What could be going on here? Recurrent acidosis without AG?

This stuff is laced with NH4Cl

We are not allowed to sleuth patients ‘ belongings or to pursue these issues further.

We hold the US CIA to be responsible for leading us to the final diagnosis.

Lüth and Luft. NDT 2006

A prophylactic pacemaker

86 year-old man comes to ER because of weakness and confusion.

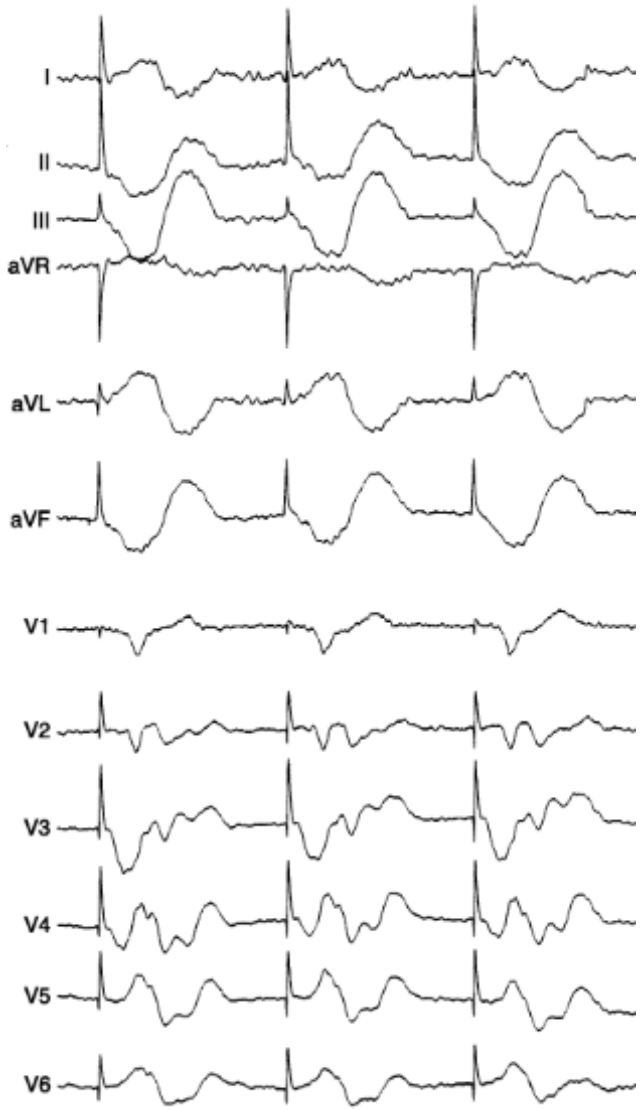
BP 75/40 mm Hg, HR 70/min
He has a lump on his left breast

Na 139, Cl 100, HCO3 10 K 11 (mmol/L)

Creatinine 4.9 mg/dL

ECG is shown

What is going on here?



Patient recently discharged with:

ACE inhibitor

Spironolactone

Beta blocker

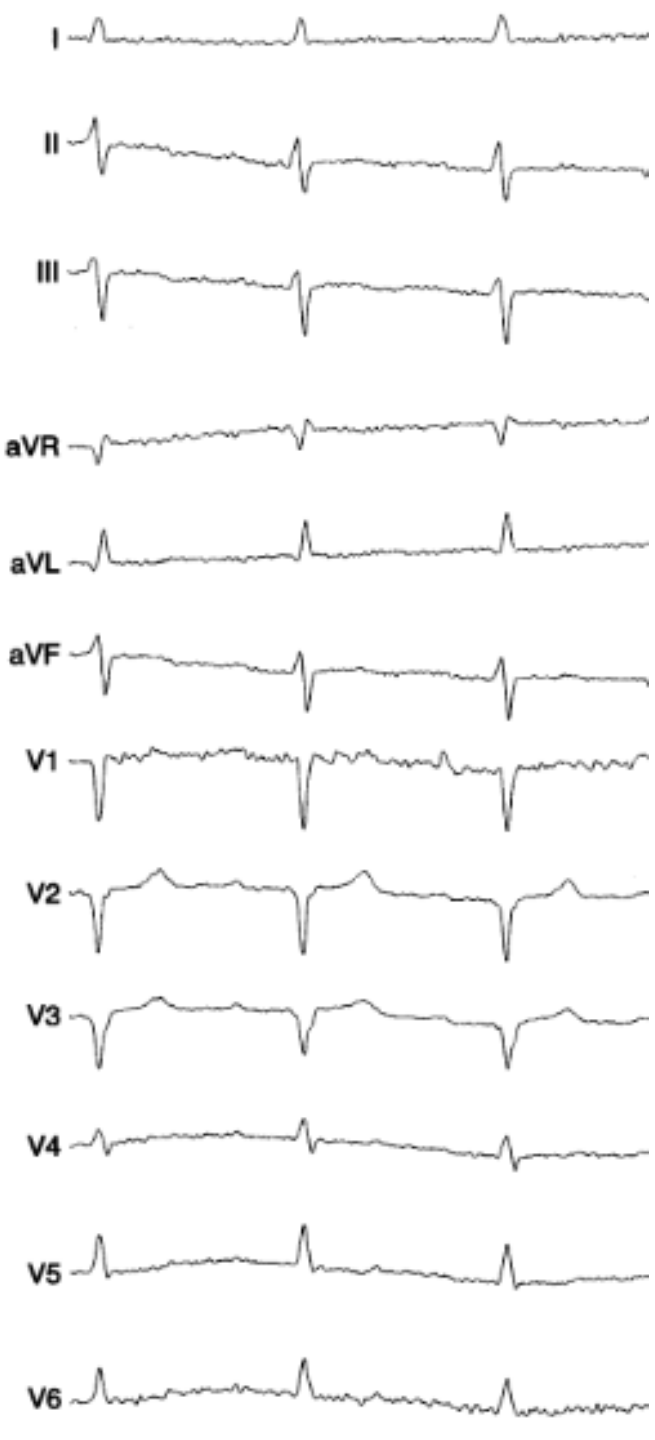
Digitalis

HCT+triamterene

With careful volume expansion and stopping these drugs, his ECG normalized

The old-fashioned „demand“ pacemaker went „back to sleep“

Stabroth et al. Am J Med 1999



Atrial fibrillation after soccer match

41 year-old Scot visits Berlin on business.

Watches Manchester United against Glasgow

Drinks three (or so) beers

He becomes extremely week (quadriparetic)

In ER, BP 154/65 mm Hg, HR 100

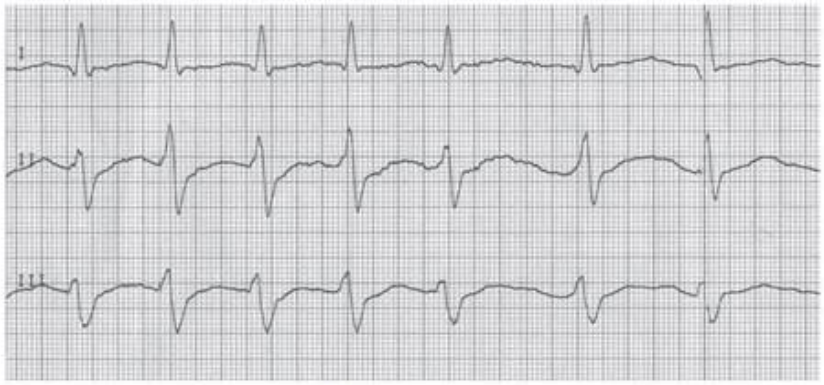
pH 7.43, PaCO2 35, PaO2 54 (mm Hg venous)

Na 140, Cl 111, HCO3 24, K 1.6 (all mmol/L)

Lactate and glucose normal.

This patient has profound hypokalemia,
But his acid-base status is normal.

What does he have?



TSH value was <0.01 µIU/L (very low)

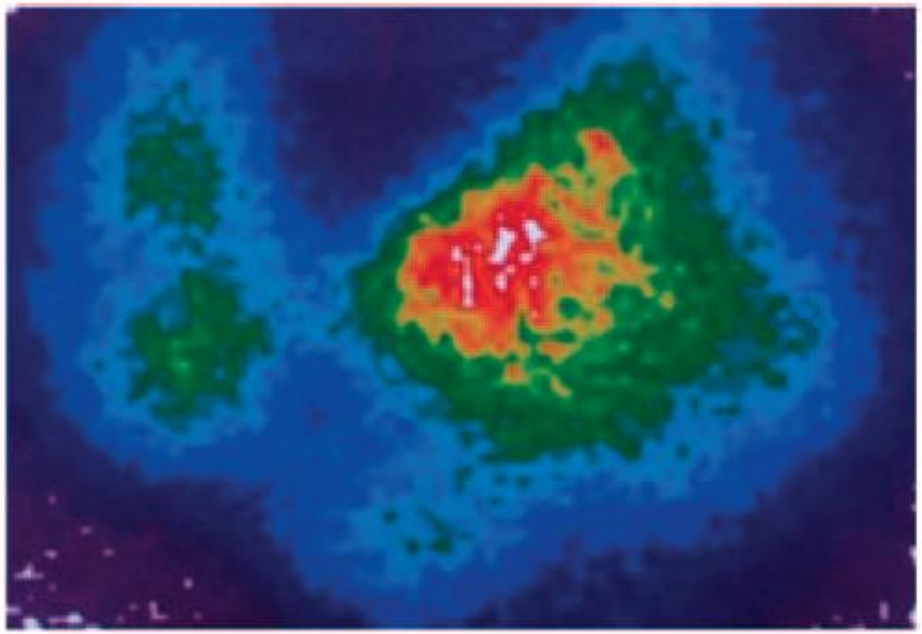
T3 17.4, T4 4 pg/dL. (T3 is 3X normal)

Serum K disturbances always feature A-B problems. If they do not – then periodic paralyses (K shfits) are possible.

Thyrotoxic periodic paralysis (TPP) is more Common amongst Orientals but does occur in Europeans.

Various „channel“-opathies can cause periodic paralysis.

Removal of this nodule cured our patient.



Polzin et al. Kidney Int 2011

The woman that „refused to respond“ to Lasix

39 year-old woman underwent laminectomy for backpain

All laboratory values were normal.

During OR, she had a fall in BP and post-operatively her PCr value was 2.5 mg/dL

She became oliguric and gained 20 kg in weight.

A „ton“ of diuretics were given – all to no avail

BP 140/90 mm Hg, HR 86/min, she was dyspneic.

A systolic murmur was attributed to tricuspid regurgitation

An echocardiogram was „hyperactive – normal“.

Electrolytes were normal, UNa was 11 and UK 24 mmol/, Uosm 533 /kg/water.

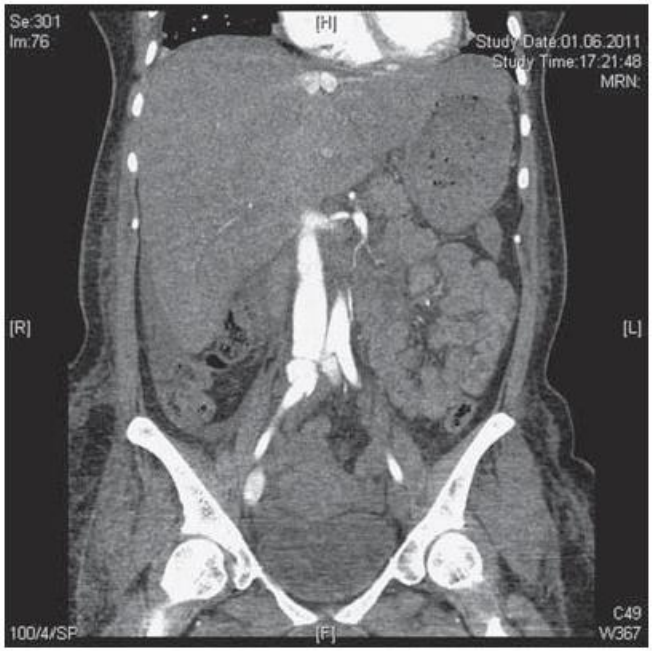


Figure 1 | The computed tomography (CT) shows that the liver is greatly enlarged; the spleen moderately so. Fluid can be seen within the abdomen. Contrast agent can be seen in the aorta to the bifurcation. Contrast immediately fills the inferior vena cava, leaving little for the renal parenchyma and lower extremities.

Table 1 | Selected laboratory values before and after stent placement

	Before stent	2 h after stent	12 h after stent
Na (mmol/l)	131	136	134
Cl (mmol/l	93	94	91
K (mmol/l)	3.8	3.7	3.4
Creatinine (mg/dl)	1.45	—	0.69
Uric acid (mg/dl)	13.5	—	7.1
Urine			
UOsm (mOsm/Kg H_2O)	318	284	308
UNa (mmol/l)	27	113	112
UK (mmol/l)	61	12	12

Vascular injury with this operation is uncommon but can occur

We call this condition: „high-output“ heart failure.

Physical diagnosis should be enough to find the problem.

„Genomic medicine“ will not help here.

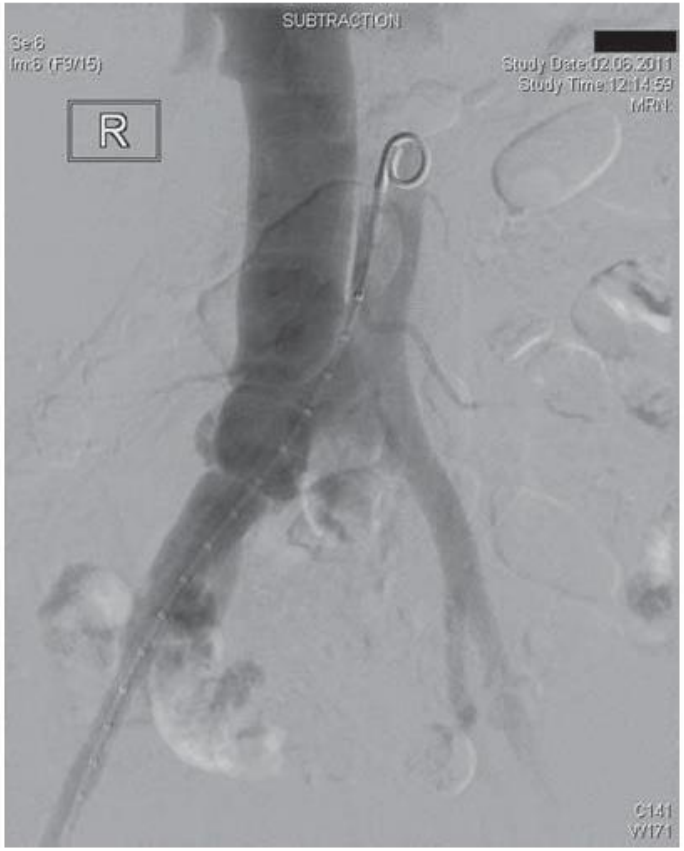


Figure 2 | Angiogram showing prompt filling of the inferior vena cava, when contrast material was injected into the distal aorta.

Flenner et al. Kidney Int 2012

Acid-base diagnoses in the 21st century

46 year-old alcoholic man on Christmas eve.

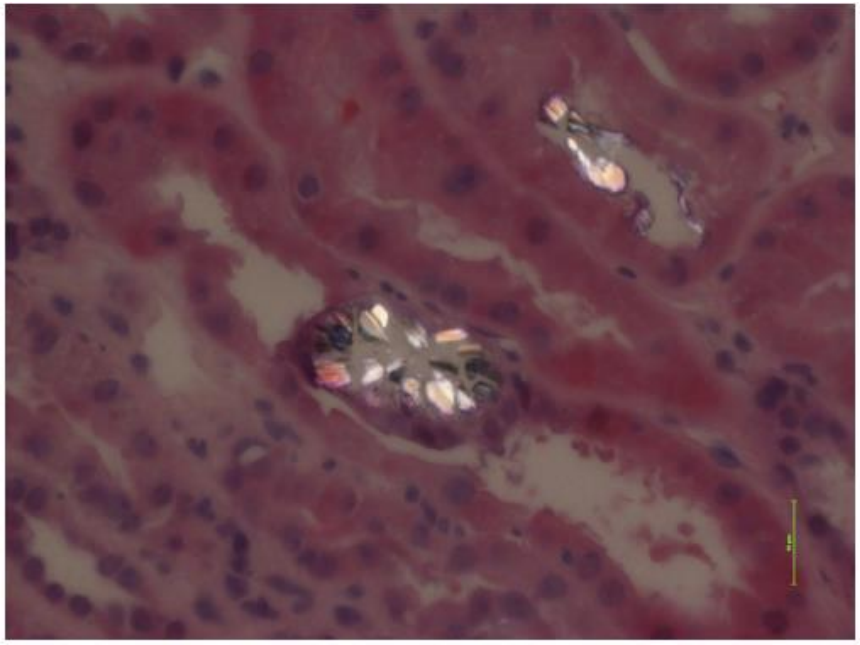
Stopped celebrating when is GCS value was 8

Urine pH was 6.0, Protein at ++, urine not examined further

pH 7.30, PaO2 114, PaCO2 23 (mm Hg)
Na 148, K 4.5, Cl 113, HCO3 9.2 (mmol/L),
„point of care“ analyzer reports lactate at 38 mmol/L

Clinical Lab later reports lactate at 11 mmol/L

What is going on here?



AG is 26 mmol/L. Lactate of 38 does not fit with an AG of 26. POC analyzers show falsely elevated Lactate values when glycolate and glyoxylic acid are present. Patient had ethylene glycol values of 1.5 mg/dL. Fomepizole was given.

Kettritz et al. Kidney Int 2017

Hard times with hard water

69 year-old man with coronary disease and renal failure

Severe heart failure was also found.

SCr 700 µMol/L, pH 7.13, HCO3 9 mmol/L, dialysis seems indicated. Calcium 1.78, Phos 2.1 (mmol/L)

Dialysis was done and patient became „comatose“

Postdialysis calcium was 5 mmol/L. We examined ECGs.

So – we performed a second hemodialysis. This procedure made him no better. Calcium stayed at 5 mmol/L.

What could cause this “malignant“ hypercalcemia?

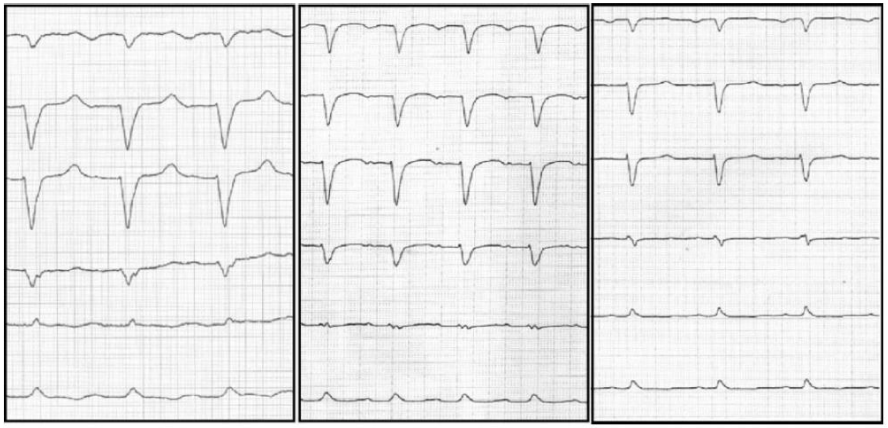


Fig. 1. ECGs obtained at different time points. Left panel: leads V1–V6 at admission. Middle panel: shortening of the ST interval and flattening of the T wave during hypercalcaemia. Right panel: normalization after calcium was returned to normal range.

Nephrologists rarely (today never) crawl on hands and knees to actually inspect dialysis Machines from behind.

Probably few of them even know how the machines work.

Dialysis water must be purified of electrolytes (like Ca and Mg) unless you live in Seattle.

We used „reverse-osmosis“ to clean our water. The RO machine worked perfectly.

So, we got down on our „hands-and-knees“ again and found:

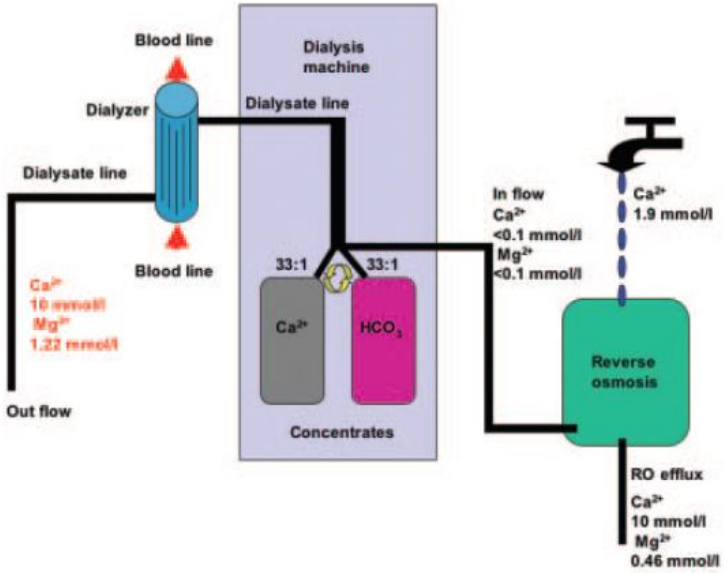


Fig. 2. Flow chart summarizing key measurements of calcium and magnesium.

Fig. 3. Reverse osmosis machine with an inlet tap (Weichwasser Zulauf) and two outlet taps, namely an efflux tap for high-calcium waste water (Konzentrat left) and an efflux tap for purified water (Permeat right) that is intended for the dialysis machine. These two taps lie next to each other and have identical connectors. The German terms Konzentrat and Permeat are confusing. It would have been better if these had been labelled 'to dialysis machine' and 'to drain'.

Such errors (language, connector, etc.) should not happen nowadays.

But nowadays is today and I am not so certain.

Nephrologists need to know the back as well as the front.

We told the family the truth what had happened and they were gracious. Patient lived at his earlier status.

I am German, but do I know what „Infusat and Permeat mean“?
Kettritz et al, NDT 2004.

Non-neurological tetraplegia

56 year-old man could not get up from couch unassisted

He was brought to our ER (surgical side)

The surgeons could not find any reason to operate so „lateraled“ him to Neurology

An 8-day workup incuding CTs, MRIs, EMGs, etc disclosed „demyelinizing polyneuropathy“

A neutritional cause was presumed and with excellent Charité food, he got better.

So, the neurologists sent him home

We could not find serum electrolytes under his many laboratory tests.

Two weeks later he is back with the same problems
Same ER also, but this time he lands on the IM side

BP 102/66 mm Hg

HR 76/min

O2 sat 97%

Cranial nerves: OK

Sensory exam: OK

Reflexes: depressed

Motor: profound upper and lower extremity weakness

Plantar responses: flexor

Substantial pedal edema

ER labs:

Parameter	Value	Reference range
Hemoglobin	15.3 g/dl	12.5–17.2
Creatinine	0.73 mg/dl	0.70–1.20
Urea nitrogen	12 mg/dl	17–48
Thyroid-stimulating hormone	2.03 mU/ml	0.27–4.0
Adrenocorticotropic hormone	20.4 pg/ml	7.2–63
Creatine kinase	1211 U/l	<190
Total protein	6.5 g/dl	6.4–8.3
Albumin	3.4 g/dl	3.5–5.2
Sodium	145 mmol/l	136–145
Chloride	111 mmol/l	98–107
Potassium	1.5 mmol/l	3.4–4.5
Phosphate	0.44 mmol/l	0.87–1.45
Magnesium	0.86 mmol/l	0.66–0.99
Urine pH	7.0	
Urine glucose	Negative	Negative
Albumin-to-creatinine ratio	13 mg/g	<20
Urine osmolarity	247 mOsm/l	
Urine sodium	65 mmol/l	
Urine potassium	15 mmol/l	
Urine phosphorus	12.2 mmol/l	
Urine creatinine	52 mg/dl	

ECG showed marked „U“ waves

pH 7.39, PaO2 92, PaCO2 24 (mm Hg)
Na 137, Cl 115, HCO3 14 (mmol/L)
By this time his K was 3.6 mmol/L

Urine pH stayed around 7.0
FENa OK

Tubular phosphate reabsorptioin stayed at 61%, despite hypophosphatemia

De Marchi et al. N Engl J Med 1993
„Renal tubular dysfunction in chronic alcohol abuse“

After 4 weeks abstinence, strength returned

Kettritz et al. Kidney Int 2016

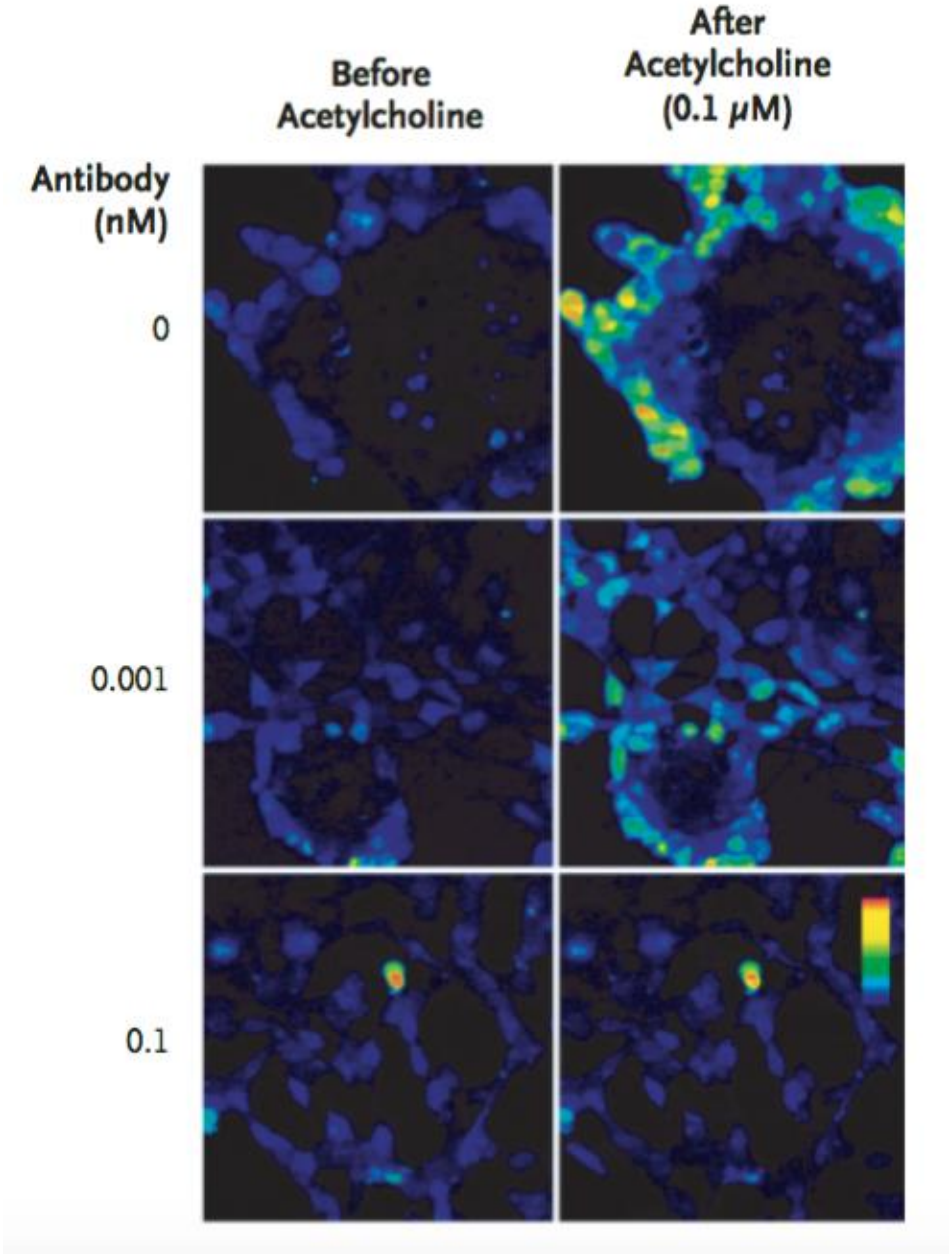
Transient proximal RTA and tubular dysfunction in alcoholism

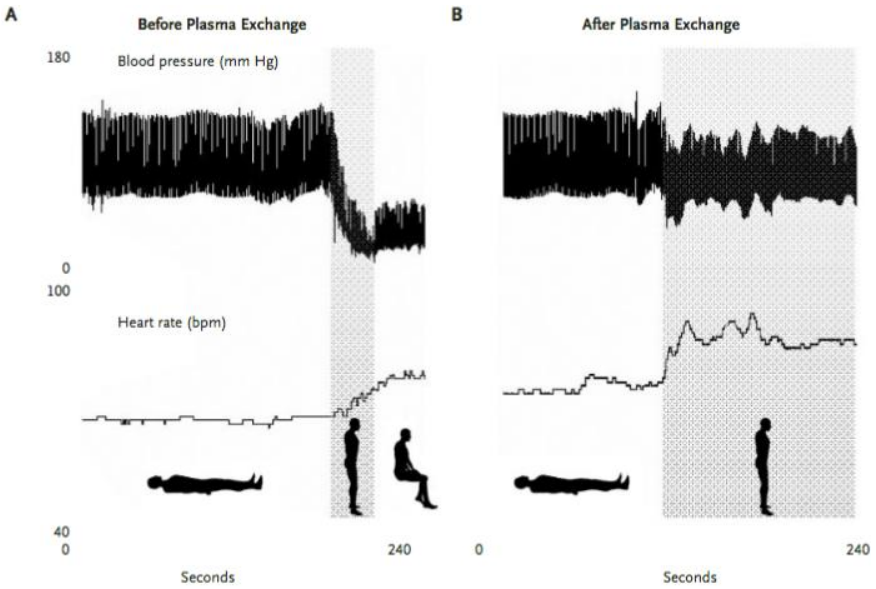
The hexamethonium man

"HE IS A PINK COMPLEXIONED PERSON, EXCEPT WHEN HE HAS STOOD for a long time, when he may get pale and faint. His handshake is warm and dry. . . . He is thin because his appetite is modest; he never feels hunger pains and his stomach never rumbles. . . . As old age comes on he will suffer from retention of urine and impotence but frequency, precipitancy, and strangury will not worry him." Paton's description of the "hexamethonium man"

1. Cannot stand without fainting (BP 88/60 mm Hg)
2. No change in heart rate
3. Constipated and received colostomy
4. Difficulty urinating
5. Impotent. Penile operation did not help
6. Pupils dilated and unresponsive to light
7. Cold pressor test no response

Primary autoimmune autonomic failure





Antibodies directed at ACh receptor subunit present on autonomic nerves

Schroeder et al. N Engl J Med 2005

Calcium, phosphorus, and clinical thought patterns

50 year-old woman would like a „second opinion“

Her problem is bone pain for several years. She also has „stress fractures“ wihtout trauma or stress.

Her doctors find a monoclonal spike (light chain) and a bone marrow aspirate finds 15% plasma cells.

Her calcium is OK, but her phosphorus is continuously low, which bothers no-one.

Imaging found „no specific osteolytic lesions“

She receives lenalidomide, bortezomib and dexamethosone.

Thereafter, high-dose phenyl-alanine-mustard and autologous stem-cell transplantation were performed.

But her complaints got no better.

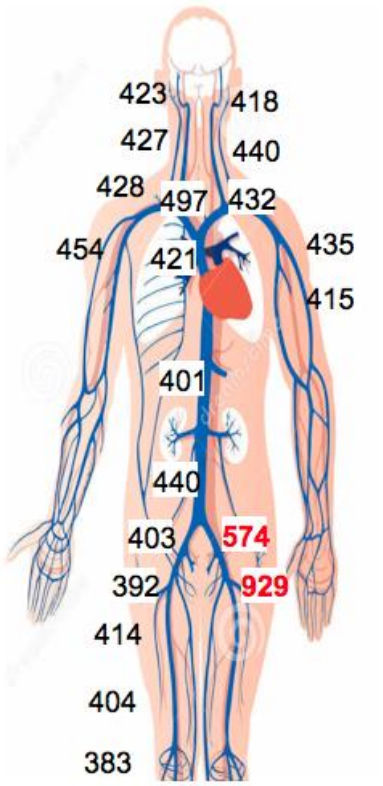
Na 141, K 3.9, Cl 102, Ca 2.4, Mg 0.76, and Phos 0.46. PTH moderately elevated, vit D normal, ALK Phos mildly elevated.

Urinary Phos (we measured it) was 11 mmol/L (Tubular Phosphate Reabsorption 53%). Should have been 100%.

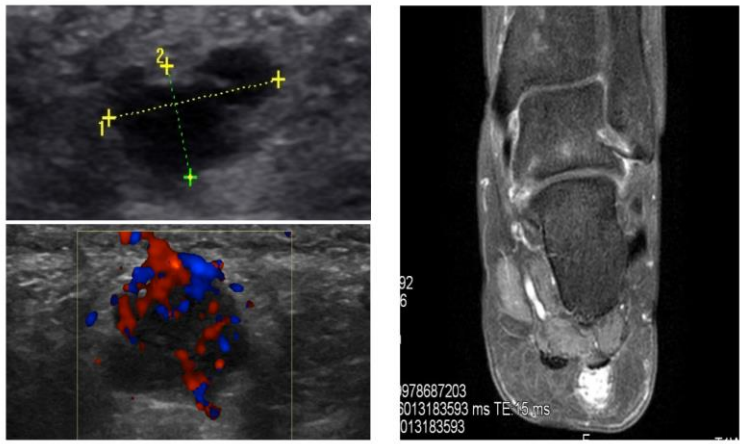
We measured FGF23

FGF-23 values

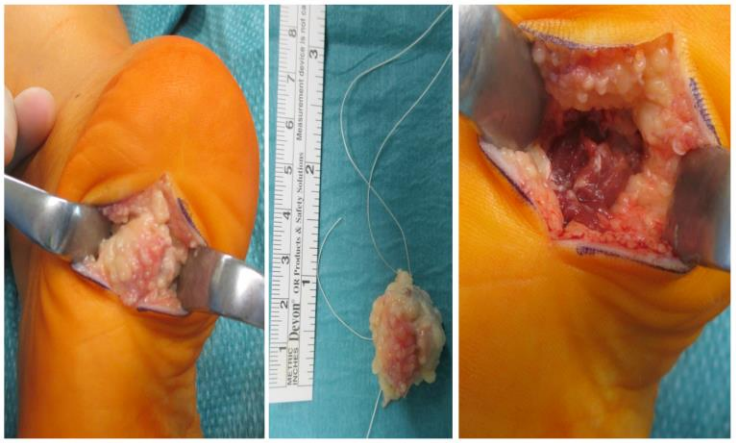
(normal 26-110 RU/L)



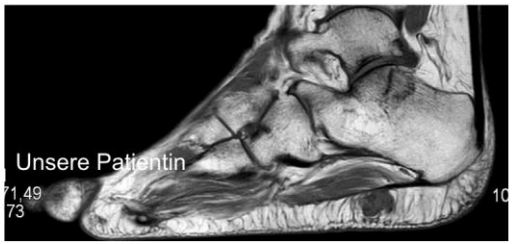
And then what?



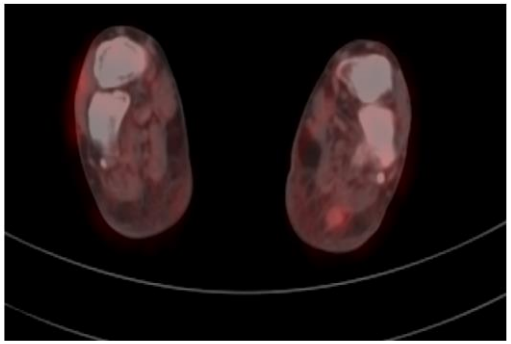
Resection and operative specimen



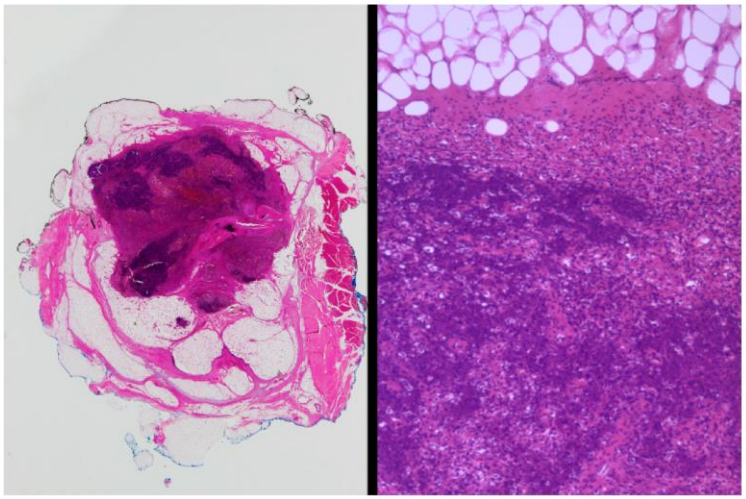
MRI



PET-CT



Hemangioma of the plantar fascia



FGF23-producing tumor